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Exploring hotspots of pneumococcal pneumonia and potential impacts of ejecta dust exposure following the Christchurch earthquakes

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ABSTRACT

The etiology of pneumococcal pneumonia (PP) is well-known. Yet, some events may increase its incidence. Natural disasters may worsen air quality, a risk factor for PP. We investigated spatial/spatio-temporal clustering of PP pre- and post-earthquakes in Christchurch, New Zealand. The earthquakes resulted in deaths, widespread damage and liquefaction ejecta (a source of air-borne dust). We tested for clusters and associations with ejecta, using 97 cases (diagnosed 10/2008–12/2011), adjusted for age and area-level deprivation. The strongest evidence to support the potential role of ejecta in clusters of PP cases was the: (1) geographic shift in the spatio-temporal cluster after deprivation adjustment to match the post-earthquake clusters and; (2) increased relative risk in the fully-adjusted post-earthquake compared to the pre-earthquake cluster. The application of spatial statistics to study PP and ejecta are novel. Further studies to assess the long-term impacts of ejecta inhalation are recommended particularly in Christchurch, where seismic activity continues.

1. Introduction

Invasive pneumococcal infections are a major global health problem, resulting in significant mortality and morbidity including pneumonia, meningitis and septicaemia. The World Health Organization estimates that more than 1.6 million people die every year from pneumococcal infections (NIAID, 2012). Pneumococcal pneumonia (PP) is caused by the *Streptococcus pneumoniae* bacterium, and is one form of invasive pneumococcal disease. PP tends to affect humans when they are either very young or very old. Risk factors include household crowding, nutrition, passive and active tobacco smoke (Zanobetti and Woodhead, 2010), heavy alcohol intake, contact with pets or infants, poor dental health and other respiratory comorbidities such as chronic bronchitis (Almirall et al., 2008). Each year in New Zealand, there is a distinct seasonal pattern with the highest number of notifications reported during winter. Also, the rates are highest in those over 70 years and those aged less than 1 year, the Pacific Peoples and Māori (ESR, 2012a), and deprived areas (using the New Zealand deprivation index or NZDep) (ESR, 2011b). This is likely due to the fact that NZDep is comprised of census variables (Crampton et al., 2004) including a known risk factor, household crowding, and the fact that NZDep is strongly correlated with smoking (Barnett et al., 2005), another risk factor, particularly for those aged less than 5 years (ESR, 2012a).

While the agent responsible and the risk factors for PP are well-established, it has been posited that air pollution (especially particulate matter) may also influence the

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development of PP (Zanobetti and Woodhead, 2010). The biological pathways by which inhalation of particulate matter may influence PP risk include damage to epithelial cells and pulmonary edema (Ferin, 1994). In addition, high doses of particles can prompt impaired cellular defense and immune system change (Kreyling et al., 2004; Oberdorster et al., 2002). In related research, patients with prolonged inhalation of silica (silicosis) have up to three times the risk of developing lung infections, including tuberculosis. It is suggested that silica damages the ability of pulmonary macrophages to kill bacteria (Cowie, 1994). Other studies have found that inhalation of inorganic dust (including quartz, dust from cement, and man-made mineral fibres) increased mortality from infectious pneumonias among construction workers (RR = 1.87) (Torén et al., 2011). In addition to damage which increases susceptibility to infection, the health relevance of the microorganisms that particles themselves carry remains largely unclear (Griffin, 2007). A recent study examined the microbial components and particle sizes of dust from storm events in the Mediterranean, detecting *Streptococcus pneumoniae* on particles ranging from 0.55 to 7.9 μm in size (Polymenakou et al., 2008). Still, given this evidence, it is unclear whether dust inhalation could be a direct cause of PP (due to bacteria on the particles) or whether dust inhalation may reduce immune-system function, leading to increased susceptibility to PP. The scant research in this area has typically evaluated the potential relationship in occupation settings and in relation to major storm events. Little is known about the potential relationship between PP and other events, such as natural disasters, which result in high levels of airborne dust.

Significant seismic activity started on 4th September 2010 in Christchurch and the Canterbury region in New Zealand. This has been followed by over 10,000 aftershocks including a major event on 22 February 2011 which directly caused nearly 200 fatalities and severely damaged over 10,000 buildings and residential dwellings (Reyners, 2011) and much of the city's infrastructure. In the history of New Zealand, this event caused unparalleled destruction (Bradley and Cubrinovski, 2011). At the time of writing, the city centre remains behind a cordon, and the entire metropolitan area is involved in active recovery. One unique feature of the earthquakes in and around Christchurch was the severity and spatial extent of liquefaction. Liquefaction occurs when saturated, loose soil loses its stiffness due to shaking. Pore water pressure builds, which pushes water and fine silt and sand through cracks in the surface of the ground. When the sand and silt material on the surface is exposed to air, it dries and easily becomes airborne (subsequently ejecta dust). Soil liquefaction repeatedly occurred at areas in the city, particularly during the 4 September 2010, 22 February 2011, and the 13 June 2011 earthquakes, magnitudes 7.1, 6.3 and 6.3, respectively (Cubrinovski, 2011). In fact, over 320,000 tonnes of ejecta was removed from the city as of 10 March 2011 (CCC, 2011). In addition, the number of high pollution nights[1] doubled in 2011 compared to previous years, a fact attributed to the wind-blown ejecta dust produced by liquefaction (ECan, 2011). A recent study conducted by New Zealand's Environmental Science and Research Limited (ESR) assessed the microbiologic content of ejecta samples collected 5 weeks after the February earthquake, testing for faecal pathogens (*Escherichia coli*, MS2 phage, enterovirus and rotavirus) (ESR, 2011a). ESR concluded that none of the faecal indicators were detected above guidelines and ejecta represented a low risk of bacterial and viral infection to the public. It is worth noting that the sample size was very small ($n = 21$) and only involved two neighbourhoods in Christchurch and did not explicitly examine for *Streptococcus pneumoniae*. It has been shown that liquefaction ejecta contained up to 65% silica and that, of the respirable fraction, about 30% was quartz (ESR, 2012b). In addition, it has been shown that liquefaction silt contains particles smaller than 10 μm (ESR, 2012b) and this contributed to the additional high pollution nights (ECan, 2011).

It is theorized that inhalation of ejecta dust may lead to increased respiratory infections directly, or may increase susceptibility to infections, including PP. However, only portions of the region were affected by ejecta. For this reason, we investigated whether there were hotspots of PP cases ooccurring before and after the onset of earthquakes and whether these hotspots coincided with areas affected by ejecta. We used spatial scan statistics to test the following null hypotheses: (1) that geographical PP case data are randomly distributed against a localised cluster alternative both before and after the earthquakes, after adjustment for age; (2) that geographical PP case data are randomly distributed against a localised cluster alternative both before and after the earthquakes after adjustment for age and deprivation; and (3) that there is a spatial clustering of cases after the earthquakes which are clustered in areas affected by severe liquefaction after adjustment for age and deprivation.

2. Study area

This study involved laboratory confirmed cases of PP notified to the medical officer of health, Canterbury District Health Board, between October 2008 and December 2011, inclusive. This study period was selected to provide spatial and temporal comparison of cases, prior to and following the onset of earthquakes (4 September 2010). The study area included Christchurch municipality census area units (CAUs) within the Canterbury region (Christchurch territorial authority), with the exclusion of Banks Peninsula (where there was one case which was omitted from the analyses). CAUs are the second smallest unit of dissemination of census data in New Zealand. There are over 1900 CAUs in New Zealand and 114 CAUs in the study area, each representing an average of 3038 people.

3. Methods

3.1. Data

Invasive pneumococcal disease has been a notifiable disease since 17 October 2008, following the introduction

[1] Defined as a 24-h average level of PM_{10} above the National Environmental Standard of 50 $\mu g/m^3$.